The Use of Thallium 201 Myocardial Imaging to Exclude Myocardial Infarction After Dissection in Congenital Coarctation of the Aorta

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The use of a mobile gamma camera with thallium 201 myocardial imaging is described to exclude myocardial infarction in a patient admitted to the coronary care unit in shock and with clinical, enzyme, and ECG changes consistent with infarction. The patient suffered from acute aortic dissection associated with congenital coarctation of the aorta. The myocardial scan excluded transmural myocardial injury.

Dissection of the aorta is amenable to surgical correction if dissection of the coronary ostia has not caused myocardial damage. Thallium-201 scintigraphy using a portable gamma camera is a noninvasive bedside method of assessing myocardial perfusion and excluding myocardial damage before embarking on extensive invasive investigation as a prelude to operative intervention. This report shows the value of thallium scintigraphy in the exclusion of significant myocardial damage in a patient with coarctation of the aorta and cardiogenic shock.

CASE REPORT

A 27-year-old healthy man was admitted to a regional hospital with a sudden, severe, retrosternal chest pain. He had been well and had not had a previous medical examination. On admission he was severely hypotensive (blood pressure, 80/60 mm Hg) but following bed rest, the blood pressure rose to 120/80 mm Hg, and the chest pain disappeared. The next day mild hypertension was noted, and a diastolic murmur was heard at the left sternal border. The ECG showed ST segment elevation in the lateral and diaphragmatic leads, and the chest x-ray film showed dilatation of the ascending aorta. A diagnosis of dissection of the aorta, possibly with severe myocardial damage, was considered. He was treated with propranolol and bed rest.

On the seventh hospital day, he complained of recurrent chest pain. The blood pressure was 160/110 mm Hg, and a sudden left hemiplegia appeared. The patient's condition deteriorated rapidly; he lapsed into severe cardiogenic shock and was transferred to the coronary care unit in our hospital for evaluation and management. The patient was stuporous and had a left hemiplegia. Arterial pulses were palpable in both carotid arteries, but not in the upper or lower limbs, and the blood pressure could not be measured. The JVP was raised to 15 cm. The cardiac apex was felt in the sixth intercostal space in the midaxillary line. The first and second heart sounds were normal. A systolic click and a short, grade 2, mid-systolic murmur were heard at the apex and at the fourth intercostal space at the left sternal border. Results of examination of the chest and abdomen were unremarkable. The ECG showed sinus rhythm, a PR interval of 0.20 sec, and ST segment elevation (Fig 1).

Chest x-ray film showed dilatation of the ascending aorta and aortic root.
brachial pulses became palpable. No pulses were found at or below the femoral arteries. A diagnosis was made of coarctation of the aorta with acute dissection of the proximal aorta, but the nature of the cardiac lesions remained uncertain. The differential diagnosis included rupture of the aorta into the pericardial space with cardiac tamponade and epicardial irritation, or dissection of the aorta involving the coronary ostia with massive myocardial infarction. A $^{201}$TI myocardial scan was performed using a portable gamma camera (Dycomat, Elscint, Ltd, Haifa, Israel) in the coronary care unit. Imaging began ten minutes after injection of 2 mCi of $^{201}$TI IV through an antecubital vein. Images were recorded in the left lateral, left oblique, and anterior projections. The scan was normal and showed normal perfusion of the myocardium; transmural myocardial infarction or damage was thus excluded (Fig 2). Aortography was performed by a percutaneous puncture of the right femoral artery. The catheter was passed into the ascending aorta, and a gradient of 60 mm Hg was measured across the coarctation, which was located at the usual site distal to the aortic arch. Aortography of the ascending aorta confirmed the presence of an aortic dissection and rupture with a leak into the pericardial space. The ascending aorta was greatly dilated (Fig 3). The catheter was passed up both carotid arteries. Following aortography, the neurologic state of the patient continued to deteriorate rapidly. Computerized axial tomography of the brain showed severe edema of the right cerebral hemisphere with marked deviation of the midline and complete obstruction of the right cerebral ventricles. The neurologic condition was regarded as irreversible and a contraindication to aortic surgery. The patient died two days following admission. Permission for autopsy could not be obtained.

**DISCUSSION**

This young man was admitted to the hospital with a likely clinical diagnosis of coarctation of the aorta and aortic dissection complicated by left hemiplegia.1 He then had profound cardiogenic shock with diffuse ST-T changes on the ECG. The nature of the ST-T changes was uncertain: were they pericardial or did they represent myocardial injury? The $^{201}$TI scintiscan was diagnostic and conclusive: it showed normal myocardial perfusion and excluded severe transmural myocardial damage. Minor damage would not explain the picture of cardiogenic shock and would be most unlikely to be the only result of coronary ostial occlusion.

The raised serum bilirubin and BUN and the extraordinary rise in SGOT were signs of extensive visceral damage owing to a low cardiac output and poor splanchnic, renal, and muscle blood flow. Prolonged hypoperfusion was responsible for minor myocardial damage and the raised CPK-MB must be attributed to subendocardial damage secondary to the profound hypovolemic shock. The ST segment elevation on the ECG is probably a result of blood in the pericardial space and pericardial irritation.

$^{201}$TI is extracted by healthy myocardium by activation of the membrane-bound sodium potassium adenosine triphosphatase, and the initial distribution of the radionuclide in the myocardium is proportional to myocardial blood flow.3-5 Myocardial infarction creates a permanent defect in the myocardial perfusion image: transient ischemia induces an initial defect, but this gradually takes up the thallium so that the "cold spot" has disappeared after a few hours.4 $^{201}$TI scintigraphy detects infarcts producing 6 percent asynergy in over 90 percent of patients but is less sensitive to small in-
farcts. It is not a reliable method of demonstrating sub-endocardial infarctions.6

In our patient, the normal scintiscan excluded important myocardial ischemia, and we believe this to be the first such patient described in the literature. The normal thallium scan excluded irreversible myocardial damage as a cause for the hypotension. It suggested that aortic dissection was the underlying lesion, that this was amenable to surgery, and that angiography was therapeutically relevant to permit accurate anatomic definition of the coarctation and site of the aortic dissection. Unfortunately, the patient's neurologic condition deteriorated, as a result of increasing cerebral edema and aqueductal obstruction: the severe irreversible neurologic damage made surgery impractical, and the patient died in deep coma.

REFERENCES

1 Levinson DC, Edmeades DT, Griffith GC. Dissecting aneurysm of the aorta: its clinical, electrocardiographic and laboratory features: report of 58 autopsied cases. Circulation 1950; 1:360-87


